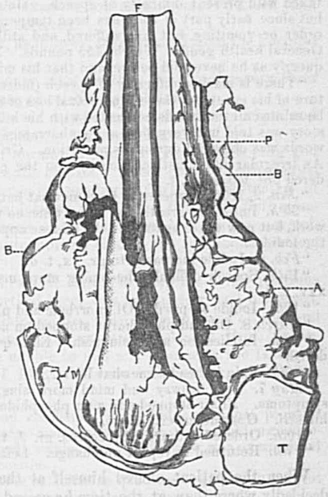
On making a longitudinal median section, involving the shaft of the femur and of the tibia, the following appearances were presented, as described by Dr. F. Delafield :—

"There is a tumour occupying five inches of the lower end of the femur; the tumour has completely replaced the medulla of the bone, and at a point four inches from the lower end of the bone has completely destroyed its shaft, so that there is an oblique fracture. The tumour has grown backwards, so as to form a circumscribed tumour the size of a goose's egg in the popliteal space. On the anterior and lateral surfaces of the femur is a periosteal tumour five inches long and two inches thick. The popliteal artery runs over the tumour in the popliteal space.

"*Microscopic Examination.*—The periosteal tumour consists of an abundant reticulated and fibrillated stroma, with a large number of cells. The cells are round, oval, fusiform, irregular, many large, some giant cells. The medulla of the shaft is replaced by new tissue, also the medulla of the cancellous bone forming the lower end of femur. This new tissue has more basement substance and fewer cells than the periosteal tumour. The shaft of the bone is destroyed by the new growth following the Haversian canals."



A. Supposed aneurism; B B. Secondary growth; P. Popliteal artery; F. Femur; T. Tibia.

Art. X.—*Partial Aphasia without Appreciable Lesion of Island of Reil.* By Horatio C. Wood, Jr., M.D., Professor of Materia Medica and Therapeutics, and Clinical Professor of Diseases of the Nervous System, in the University of Pennsylvania.

James D——, æt. 33, presented himself at the University Hospital Dispensary, June 11, 1875. He stated that he had been for a long time under treatment at the Orthopædic Hospital, without considerable improvement, but had discontinued his visits there for some time. Subsequently a history of his case was kindly furnished by Dr. Geo. S. Gerhard of the Orthopædic Hospital staff; this is here inserted instead of the crude and very imperfect account given by the patient's friends.

"Was admitted as out-patient Oct. 28, 1874. Served in army during war in infantry, and was once slightly wounded. He never had any symptoms of lead poisoning, but had chancre in 1863. He never had any secondaries, and was always perfectly well until two months ago (end of July), when he was suddenly taken with present difficulty of speech. He was formerly addicted to sprees, but since early part of July has been temperate. He has not had vision disorder or vomiting, but has suffered, and still suffers, from frontal headache. General health good. Weight 155 pounds. Mother says he is silly, and acts queerly as he never did before, and that his mind is feeble.

"There is marked difficulty of speech (indeed this is the most prominent feature of his case), but there is no actual loss of memory for words. For instance, he related all the details connected with his killing a snake ten years ago. The story was told in a very slow and embarrassed way, but no loss of memory for words was observed during its narration. Grip of right hand feebler than left. An irregular and indistinct blue line on the gums. Eye ground normal. Ordered pot. iod. gr. x, t. d.

"*Jan.* 2, 1875. Appears to be somewhat better. Continue treatment.

"25*th*. Improved greatly for some time, so that he was able to resume his work, but now worse again, and the relapse appeared to follow neglect in taking the iodide.

"*Feb.* 8. Iodide increased to gr. xx, t. d.

"15*th*. Speech difficulty becoming more marked. Iodide increased to gr. xxv, t. d.

"19*th*. Iodide stopped. Ol. morrhuæ and phosphorus gr. $\frac{1}{16}$ t. d. ordered.

"*March* 8. Oil and phosphorus stopped on account of causing nausea.

"19*th*. Intellection more sluggish. Elix. quin., strych. et ferri, f℥j t. d. ordered.

"29*th*. Pain in head somewhat better.

"*May* 7. Very drowsy and mind more sluggish than ever. No paralytic symptoms. Elixir stopped and zinc phosphide ordered. Able to take care of himself. Goes about city alone.

"10*th*. Ordered hydrarg. chl. corros. gr. $\frac{1}{12}$ t. d. Zinc stopped.

"17*th*. Returned to report no change. Left off attendance."

When the patient offered himself at the University Hospital he was decidedly worse than at the time he ceased his visits at the Orthopædic Hospital. There was not, however, any distinct paralysis; there was general muscular weakness, but not more than is often seen in chronic disorders; the chief complaints were of a persistent severe frontal headache, with marked paroxysmal exacerbations. There had been no fits, but there was a very decided, although not complete, aphasia. The patient was entirely unable to give any account of himself, or to frame any connected sentences. When asked whether he had ever had the "bad disease," he answered with great slowness and effort "war—rotted me—bitch;" meaning that he had got the disease from a woman whilst in the army. The special senses were unimpaired. Ophthalmoscopic examination showed complete absence of choked disk or descending neuritis; there was some neuroretinitis, which was pronounced by Dr. Wm. F. Norris to be probably the result of the existing high degree of myopia.

Large doses of iodide of potassium were prescribed and the patient sent home. That evening, or the next one, he was seized with violent convulsions, with delirium and unconsciousness. A neighbouring practitioner, having been called in, pronounced the case one of strychnia poisoning, and treated it as such for four days, when I was called in. At this time there was furious delirium, with constant screaming and fighting, but no convulsions. The case was pronounced meningitis, and a fatal prognosis given. The man was sent on the 18th to the hospital, where, under active blistering, the free use of calomel and bromide of potassium, he got better, but never became rational. About 11 o'clock in the morning of the 29th, he fell into a sleep, which deepened into a stupor, in which he died about four o'clock in the next afternoon.

His pupils were widely dilated and immovable during his stay at the hospital, but on ophthalmoscopic examination nothing more decided than some retinal congestion could be detected.

The notes of the post-mortem, as follows, are extracted from the hospital register :—

"Examination made eight hours after death : *Dura mater* very thick and opaque. Very great increase of cerebro-spinal fluid.

"*Arachnoid* closely adherent over upper surface of brain, opaque and thick with spots of very great thickness here and there.

"Consistency of *brain* about normal to touch. Great thickening and exudation of lymph behind the optic chiasm. Lobes closely agglutinated by exudation. Left side externally seemingly normal. Softening of extreme anterior apex of middle lobe on right side. *Island of Reil* seemingly normal, though membranes over it thickened. *Lungs* normal. *Heart* healthy. A very large ante-mortem clot in the pulmonary artery, also one in left ventricle."

The brain was put in alcohol, which failed to keep the inner portions of the organ. The exterior cortical layer was fortunately well preserved, and good sections of the left Island of Reil were made in the Pathological Laboratory of the University, under the superintendence of Prof. Tyson. Prof. Tyson stated that he was unable to find any disease in the Island of Reil, and a section sent to Prof. Fitz, of Harvard University, elicited the following note :—

"Your section arrived in good condition. I found occasional pigment granules in the perivascular spaces—a rare glistening round or oblong homogeneous body suggestive of a corpus amylaceum. Also rare, was an apparent increase of nuclei in certain adventitiæ.

"None of these appearances were sufficient in quantity or quality to suggest disorganization of the cerebral substance in the respective section."

Remarks.—The chief interest of the case just narrated centres in two points, the absence of choked disk with general and local meningitis, and the presence of aphasia, without detectable alteration of the Island of Reil. The diagnostic importance of choked disk, so far as my experience goes, is not supreme. I have seen what was apparently it in a patient in whom no other distinct evidences of brain lesion could be found during a scrutiny extending over several months, and I have generally found it absent in such cases of brain tumour as presented any diagnostic difficulties, the diagnosis in one of these cases being confirmed by an autopsy, made two days after the ophthalmoscopic examination by Prof. Norris and myself. I do not mean to deny the usual diagnostic import of choked disk when present, but it seems to me in some cases impossible to distinguish it from ascending neuritis, and its absence appears to be of no value in proving the non-existence of cerebral growths, or even of meningitis.

The controversy as to the localization of function rages so fiercely that the present case will probably be seized upon at once. I have therefore had the matter carefully verified, and it seems to me thoroughly established that there was no appreciable lesion of the Island of Reil. This does not, however, to my mind disprove the theory of speech localization. The aphasia, according to the statement of the friends of the patient, had come on gradually. It was far from complete, although very marked. It is well known that the Island of Reil receives much of its blood supply from the superjacent meninges. These meninges were very much thickened, and it is perfectly possible that there should have been an interruption of circulation sufficient to interfere with the function of the part, but not sufficient to alter its structure in a degree to be appreciated by our present methods of examination.

ART. XI.—*The Relations of Blepharitis Ciliaris to Ametropia.*[1] By D. B. ST. JOHN ROOSA, M.D., Professor of Ophthalmology and Otology in the University of the City of New York, Surgeon to the Manhattan Eye and Ear Hospital.

IT is a well-recognized fact that certain forms of conjunctival inflammation arise from uncorrected errors of refraction. I do not think it is generally conceded, however, that blepharitis ciliaris often stands in the same relation to ametropia. The principal text-books do not give any prominence to the subject either in the discussion of blepharitis or ametropia. Most, if not all of them, are silent upon the subject. Donders does not, I think, even allude to blepharitis as one of the results of uncorrected strain of the accommodation. In the chapter on Blepharitis in Saemesch's Hand-Buch, by Professor Michel, the subject is not mentioned. Schweigger in his hand-book is also silent upon the point. The same may be said of the treatises of Wecker, Stellwag, and Soelberg Wells. I mention these facts because in speaking of the causal connection of blepharitis with ametropia to some of my professional friends, I found them under the impression that the subject had already been distinctly enough mentioned in the text-books. However much may have been known and said upon the subject in the practice of eye infirmaries, very little has as yet found its way into the literature of ophthalmology.

I therefore present a few statistics as to the connection between diseases of the hair follicles and tarsal glands and the various forms of ametropia. They are all the cases observed by me in private practice during the last eighteen months. I have attempted to keep a similar record in the Manhattan Eye and Ear Hospital, but there are some omissions in these statistics

---

[1] Read before the International Congress of Ophthalmology, September, 1876.